UNDERSTANDING LEAD UPTAKE AND EFFECTS ACROSS SPECIES LINES: A CONSERVATION MEDICINE BASED APPROACH

MARK A. POKRAS AND MICHELLE R. KNEELAND

Center for Conservation Medicine, Tufts University Cummings School of Veterinary Medicine, 200 Westboro Rd., North Grafton, MA 01536, USA.
E-mail: mark.pokras@tufts.edu

ABSTRACT.—Conservation medicine examines the linkages among the health of people, animals and the environment. Few issues illustrate this approach better than an examination of lead (Pb) toxicity. We briefly review the current state of knowledge on the toxicity of lead and its effects on wildlife, humans, and domestic animals.

Lead is cheap and there is a long tradition of its use. But the toxic effects of Pb have also been recognized for centuries. As a result, western societies have greatly reduced many traditional uses of Pb, including many paints, gasoline and solders because of threats to the health of humans and the environment. Legislation in several countries has eliminated the use of lead shot for hunting waterfowl. Despite these advances, a great many Pb products continue to be readily available. Conservationists recognize that hunting, angling and shooting sports deposit thousands of tons of Pb into the environment each year.

Because of our concerns for human health and over 100 years of focused research, we know the most about lead poisoning in people. Even today, our knowledge of the long-term sublethal effects of Pb on human health continues to grow dramatically. Our knowledge about lead poisoning in domestic animals is significantly less. For wild animals, our understanding of lead poisoning is roughly where our knowledge about humans was in the mid-1800s when Tanquerel Des Planches made his famous medical observations (Tanquerel Des Planches 1850).

From an evolutionary perspective, physiological processes affected by lead are well conserved. Thus, scientists are able to use rodents and fish to understand how lead works in people. Similarly, those of us interested in safeguarding wildlife health should consider humans as excellent models for lead's chronic and sublethal effects.

Given what we are learning about the many toxic effects of this heavy metal, there is every reason to switch to non-toxic alternatives. To accomplish this, a broad, cross-species ecological vision is important. All interest groups must work together to find safe alternatives, to develop new educational and policy initiatives, to eliminate most current uses of Pb, and to clean up existing problems. *Received 25 August 2008, accepted 21 November 2008.*

POKRAS, M. A., AND M. R. KNEELAND. 2009. Understanding lead uptake and effects across species lines: A conservation medicine approach. *In* R. T. Watson, M. Fuller, M. Pokras, and W. G. Hunt (Eds.). Ingestion of Lead from Spent Ammunition: Implications for Wildlife and Humans. The Peregrine Fund, Boise, Idaho, USA. DOI 10.4080/ilsa.2009.0101

Key words: Lead, conservation medicine, sentinel, public health, stakeholders.

IN 1987 A BIOLOGIST from New Hampshire brought us a dead Common Loon (*Gavia immer*). As wildlife veterinarians interested in conservation issues, we agreed to run a few tests and perform a necropsy on the loon. Examining the cadaver, the bird was found to be in good condition with perfect breeding plumage and no indications of parasites or disease. The only interesting piece of evidence was a radiographic image revealing a metal object within the bird's gizzard. During the necropsy, we were surprised to discover it was actually a lead (Pb) fishing sinker. We were even more intrigued when analysis of a liver sample concluded that the loon had died of lead poisoning. Inspired by this case, we have spent the past 20 years working on the issue of lead poisoning, examining well over a thousand dead loons and testing many other species for lead toxicosis. Many conservation and health professionals now are beginning to realize the extent to which accumulation of this toxic material has been underestimated in terms of its impact on human and animal health.

This paper will focus on a general review of a number of issues including:

- What is lead?
- How is lead handled inside animals' bodies?
- What are some of the short-term and chronic effects of lead?
- Are humans significantly different from other vertebrate animals in the ways in which our bodies handle lead?
- The importance of interdisciplinary, multispecies approaches for understanding the magnitude and threats posed by environmental lead.
- Do we really need more science to prove that lead is toxic?

WHAT DO WE KNOW ABOUT LEAD?

Lead is an element and a metal (atomic number 82). It is soft, has a low melting point (327.5 °C), a high density (11.34 g/cm^3) and is found naturally in a variety of minerals including galena, cerussite and anglesite. Unlike many natural elements, lead is not known to be required by any living organism. So what does lead poisoning do, and how do we know if an animal has lead poisoning?

Lead poisoning can have rapid, acute effects or chronic, long-term effects in people and other animals. Because of societal concerns for human health and over 100 years of focused research, we know the most about lead poisoning in people. Our knowledge about lead poisoning in domestic animals is significantly less. For wild animals, our understanding of lead poisoning is roughly where our knowledge about humans was in 1848 when Tanquerel Des Planches made his famous medical observations. We know a great deal about the more obvious cases of death and debility caused by lead, but extremely little about the more subtle, chronic, sublethal effects.

Acute and subacute effects are typically caused by relatively large doses of lead over a short period—often days to months. These effects can be dramatic and include sudden death, severe abdominal cramps, anemia, ataxia, strange headaches, and behavioral changes, such as irritability and appetite loss. Of course these signs are fairly non-specific and can be caused by many things besides lead. Chronic effects are most often the result of smaller amounts of lead being taken in over longer times – months to years. These effects can be quite subtle and nonspecific, but include all body systems. A brief list of effects documented in people includes such effects as lowered sex drive, decreased fertility (in males and females), miscarriages and premature births, learning problems, hypertension, cardiovascular disease, increased aggression and kidney problems.

We know many of the ways in which lead kills wildlife over a few weeks or months, but almost nothing of the chronic, low level effects that probably harm a great many more animals and upset ecosystem functions over time. From the perspective of conservation and ecology, this is quite frustrating. But the good news is that a great many of the physiological mechanisms by which lead acts upon bodily processes are well conserved among vertebrate species. Many references cite the fact that whether we are talking about people, condors or fish, the body handles lead in much the same way as calcium. Calcium is a crucial element for living things, being used in a wide variety of metabolic activities, signaling pathways and structural com-

pounds. This has meant that understanding how lead (Pb) works in laboratory rodents and fish has helped us understand the chemistry and physiology of lead in people. Conversely, it also means that much of what we've learned about chronic, sub-lethal effects in people can reasonably be extrapolated to non-human animals. We often think of animals as sentinels for human health; but here it is we, the humans, who are the white mice. Understanding how lead works in *Homo sapiens* can play a significant role in protecting other species and environmental processes.

In all kinds of adult animals, most lead is absorbed through the digestive and respiratory systems. Under some circumstances, primarily occupational exposures, certain forms of lead (usually the more lipophilic ones) can also be absorbed through the skin. The key is that however it enters the body, the most important step is the absorption of lead into the bloodstream. Nearly all lead vapors getting into the lungs cross into the blood quickly and easily. Inhaled small particulate matter is coughed up and swallowed. Lead entering the digestive system is acted upon by stomach acids and made into soluble salts that can be absorbed by the intestine. In adult male people about 10–15% of ingested lead is usually absorbed, the rest leaves the body in feces. But in young children, up to 50% of ingested lead is absorbed. In all age groups, absorption can be increased by conditions that stress the body including pregnancy, injury and disease.

In adult people, lead remains in the blood stream for roughly 2 weeks. During that time some of the lead is excreted from the body, but much of it begins to be deposited in the soft tissues of the body, including the liver and kidneys. Residence time in these soft tissues depends on a great many variables, but is usually on the order of a few weeks to several months. The endpoint for most of the lead in the body is the skeleton. As lead leaves most of the body's other tissues some is excreted, but much is bound into the structure of bone (again, following calcium). The half-life of bone lead is on the order of decades. This means that the skeleton serves as a source pool from which low levels of lead are mobilized back into the bloodstream as the bones remodel throughout life.

In the body, lead does many things (Needleman 1991, Casas & Sordo 2006, Ahameda and Siddiqui 2007, Diertert et al. 2007). It can bind important enzymes (primarily at their sulfhydryl groups) and inactivate them. Lead (Pb) can also displace biologically important metals, such as calcium, zinc and magnesium, interfering with a variety of the body's chemical reactions (Tables 1, 2, and 3). Lead toxicity affects all organ systems, but the most profound effects are seen in the nervous, digestive, and circulatory systems. Every time nerves transmit messages around the body, calcium is required. Thus lead can interfere with functions dependent on nerve conduction such as learning, blood pressure, reaction time and muscle contraction. Contractions of smooth muscle (peristalsis) are required to move food through the esophagus, stomach and intestines. Lead can upset this ordered contraction leading to a great deal of stomach and abdominal pain, long referred to as "lead colic" in people. In the blood stream, lead interferes with the functions of hemoglobin, limiting the amount of oxygen that is carried to organs. Lead also interrupts the formation of new red blood cells in the bone marrow, leading to anemia. Effects in the skeletal and reproductive systems can cause problems such as stunted growth and infertility (in both genders). In situations where the body needs to use bone calcium stores, like growth, fracture healing, egg shell formation (in reptiles and birds), dietary imbalances, pregnancy and lactation (in mammals), and or bone loss due to aging or osteoporosis, lead is released from bones and can cause chronic, low level poisoning.

As we have learned more and more about the sublethal effects of lead in people over the last 40 or 50 years, government agencies have acted to lower the levels of blood lead that have been considered "safe." In 1968 a level of 80 µg/dL (micrograms per deciliter) in blood was considered the level of concern for children. But as we have learned more and more about the significant effects that even low levels of lead can have, these numbers have gradually been lowered. In the 1980s the limit was set at 50 µg/dL. That was changed to 30 µg/dL in 1995, then to the current 10 µg/dL. Recent studies have shown that even levels below 2 µg/dL can cause significant, long-term effects in children (Lanphear

Table 1. Mechanisms of Pb toxicity (Needleman 1991, Casas and Sordo 2006, Ahameda and Siddiqui 2007).

- Substitutes for and competes with Ca^{++}
- Disrupts Ca^{++} homeostasis
- Binds with sulfhydryl groups
- Stimulates release of Ca^{++} from mitochondria
- Damages mitochondria and mitochondrial membranes
- Substitutes for Zn in zinc mediated processes
- Increases oxidative stress
- Inhibits anti-oxidative enzymes
- Alters lipid metabolism

Table 2. Possible mechanisms by which Pb induces neurologic effects (Needleman 1991, Casas and Sordo 2006, Ahameda and Siddiqui 2007).

- Increase in affinity for Ca^{++} binding sites
- Disrupts Ca^{++} metabolism
- Substitutes for Ca^{++} in Ca/Na ATP pump
- Blocks uptake of Ca^{++} into mitochondria and endoplasmic reticula
- Interference with neural cell adhesion
- Impairment of cell to cell connections
- Alters some neurotransmitter function
- Activates protein kinase C
- Alters Ca^{++} mediated apoptosis

Table 3. Results of lead toxicity (Needleman 1991, Casas and Sordo 2006, Ahameda and Siddiqui 2007).

- Abnormal myelin formation
- Altered neurotransmitter density
- Altered neurotransmitter release
- Increase in lipid peroxidation
- Impaired heme biosynthesis leads to anemia
- Decreased cellular energy metabolism
- Altered apoptosis

2000, Canfield et al. 2003). In some areas of the USA blood lead testing is required for children entering kindergarten. This is not a very expensive test and only requires a couple of drops of blood, and yet it is still not generally required nationwide.

Detecting lead poisoning in people or other animals is done in a variety of ways. Radiographs (x-rays) can detect metal densities and their locations in the body, but cannot tell specifically if the metal is Pb. For that, we would have to remove and test the metallic object or look at lead levels in either blood or body tissues like liver, kidney or bone. In living animals, we usually look at blood samples. We can analyze blood either for the metal itself, or we can analyze blood for the levels of some enzymes which are affected by lead, typically ALAD or zinc protoporphyrin. If the person or animal is deceased, investigators typically send samples of liver or kidney to toxicologic laboratories to measure Pb levels. As will be reviewed in other presentations, determination of lead isotopes can give us important information about the origin of lead found in people and other species.

If we think about the mining, manufacturing and recycling of lead products in the USA, it is apparent that state and federal regulatory agencies try to minimize the amounts of lead that are released into the environment. State and federal permits are required for any sort of industrial lead release. But in recreational sporting goods, we have a whole class of products used for the shooting sports, hunting and fishing which, when used as intended, end up in the environment. It is difficult to come up with exact figures for how much lead is released through these activities. According to estimates from the US Geological Survey, roughly 10% of all the lead produced in the USA or imported goes for such sporting purposes. This amounts to approximately 6–10 thousand tons of lead being released into the environment annually in the USA.

Given that we have known about lead's toxicity for a very long time, you might reasonably ask why we are still using lead for so many purposes. Several possibilities come to mind. One is simply traditional practice. We have known about and used lead for a very long time, in fact the Latin name for lead, *plumbum,* is incorporated into quite a few modern

terms including "plumber" (from the people who originally worked with lead pipes), "plumb bob" (for a lead weight on the end of a string) or "plumb stupid" to comment on the behavior of people who have had too much contact with lead. In following the early settlement patterns of the USA, it is interesting to note how many towns have names like Leadville or Galena. One reason we have used lead for so long is that it has a low melting point (621.43 °F). This means that no elaborate equipment is needed to extract the metal from rock. In fact it was probably just by noticing that a pure silvery/grey metal was left on the ground after campfires that early humans first figured out how to smelt metals, thus leading us from the stone age into the age of metals. The low melting point also means that one can melt lead and cast it into a variety of forms using reasonably inexpensive equipment. This can encourage many people to melt lead at home for making things like stained glass, fishing sinkers or lead soldiers, without using proper safeguards. Lead is quite dense, about 11 times heavier than an equal volume of water. This density is one of the characteristics that contributes to its usefulness for bullets, weights of various sorts and x-ray shielding. The fact that lead is soft, easy to work and resistant to many forms of corrosion also makes it attractive for a wide variety of uses. Last, lead has historically been fairly inexpensive; until recently only a few hundred dollars per ton. That price seems to be increasing rapidly due to new demands for lead to use in storage batteries. In addition, prices are rising because we have nearly exhausted many of the easy to obtain sources of ore and new mines are more expensive. Finally, in developed countries, new regulations to protect the health of miners, manufacturing workers, recyclers and the environment are increasing all the costs associated with this metal. Such new regulations certainly have beneficial effects for workers in the USA and other first world nations. But in this age of globalization, the rising costs of mining and production have driven many international corporations to move their activities to developing countries. In such locations there is frequently little environmental regulation, few protections for workers and little enforcement. Rather than solve the environmental and health problems inherent in making and using lead products, some of our industries have simply exported them.

Despite its utility, lead is toxic. The US Centers for Disease Control and Prevention states on its lead poisoning website: "Any combination of GI complaints, neurologic dysfunction and anemia should prompt a search for heavy metals toxicity." The federal agency charged with protecting workers' health, the Occupational Safety and Health Administration (OSHA), recommends on their website (http://www.osha.gov/SLTC/lead/) that all of us should:

- Avoid purchasing or using products known to contain lead
- Avoid inhaling dusts or fumes of lead or lead-containing compounds
- Avoid consuming food or beverages or putting items in the mouth in areas where lead-based compounds or lead-based materials are in use
- Wash hands with soap and water after handling lead.

Although we are still struggling to understand the full scope of the issue, lead poisoning is not a new problem. The toxic effects of lead have been reported for over 2000 years in both humans and animals (Nriagu 1983). Grinnell (1894) published the first USA report of lead poisoning in ducks caused by ingestion of spent shot more than 100 years ago. One of the most comprehensive clinical descriptions of human lead poisoning was undertaken by Tanquerel des Planches in 1848. Dr. Alice Hamilton, in originating the field of occupational health, performed extensive work on the social costs of industrial lead poisoning in the early 1900s (Sicherman 2003). Yet it took more than a half-century and the 1962 publication of Rachel Carson's momentous Silent Spring to refocus attention on the links between chemicals in the environment and the health of people and animals. That attention helped to catalyze a new generation of scientists to focus on lead's toxic legacy. In the public health arena, the work of Clair Patterson, Herbert Needleman, Ellen Silbergeld, Philip Landrigan and many others finally brought about the elimination of lead from most house paints and gasoline. While in the wildlife world, the efforts of a great many biologists (some of whom attended this meeting) brought about regulatory changes for hunting waterfowl, forcing the replacement of toxic lead shot with safer alternatives (Anderson and Havera

1989). Yet even today Hu et al. (2007) can state, "In the world of environmental health and environmental medicine, lead exposure remains one of the most important problems in terms of prevalence of exposure and public health impact." We have to ask ourselves why more progress to eliminate this persistent health threat has not been made.

A major impediment to progress is the disciplinary separation that has long existed among groups investigating issues related to lead poisoning. While there are multiple organizations currently working on the problem, most efforts are narrowly focused on one particular aspect of lead poisoning. We need a conservation medicine-based approach to the lead poisoning problem that overcomes barriers between the disciplines of human and animal health, barriers within the field of animal health, and barriers between researchers and the general public in order to finally eliminate this persistent health threat.

BRIDGING THE HUMAN/ANIMAL DIVIDE

While the concept of using animals as sentinels of human health is not new (Winter 2001), wildlife professionals seldom realize the wealth of information that can be gained by taking the opposite approach and using humans as sentinels for animal and environmental health. A representative medium size mammal, *Homo sapiens* is by far the best understood and most widely studied species on the planet, so why not utilize this abundance of data to help us understand our non-human counterparts? In fact, it is from the human literature that we get some of the best measures for sublethal effects of lead toxicity. Low level lead exposure has been associated with a wide range of conditions in humans, including cognitive deficiencies in children, renal impairment, hypertension, cataracts, and reproductive problems such as miscarriage, stillbirth, and decreased fertility in men and women (Patrick 2006).

Deficits in cognitive and academic skills have been reported in children with blood lead concentrations lower than 5 μg/dL (Lanphear et al. 2000). Another study found that a net increase of 1 μg/dL in the lifetime average blood lead level was correlated with a loss of 0.46 IQ points (Canfield et al. 2003). In light of these findings in humans, the cognitive effects of sublethal lead poisoning are beginning to be studied in wildlife. In Herring Gull (*Larus argentatus*) chicks, for example, effects on locomotion, food begging, feeding, treadmill learning, thermoregulation, and individual recognition were observed in nestlings dosed with lead to produce feather lead concentrations equivalent to those found in wild gulls (Burger and Gochfeld 2005). Several studies have found an association between sub-clinical lead toxicosis and delinquent, antisocial, and aggressive behaviors in humans (Sciarillo 1992, Needleman et al. 1996, Nevin 2000, Dietrich et al. 2001). Similarly, the development of aggressive behaviors has been documented in domestic dogs and cats with elevated blood lead levels, as well as rodents and songbirds (Koh 1985, Burright et al. 1989, Hahn et al. 1991, Delville 1999, Janssens et al. 2003, Li et al. 2003). Pattee and Pain (2003) document an increasing use of lead worldwide and state that "lead concentrations in many living organisms may be approaching thresholds of toxicity for the adverse effects of lead." Environmental lead exposure at low levels could very well be contributing to wildlife mortality by hindering the complex mental processes and social behaviors required for reproductive success and survival.

Abdominal pain and peripheral neuropathy are two symptoms of lead poisoning that have been described in human literature for centuries (Tanquerel des Planches 1850). Commonly referred to as "painter's colic" and "wrist drop" respectively, these conditions are not specific to humans. Abdominal pain is recognized as a clinical sign common to nearly all lead poisoned animals (Osweiler 1996). Sileo and Fefer (1987) observed "droop wing," the avian equivalent of human wrist drop, in Laysan Albatross (*Diomedea immutabilis*) fledglings that had ingested lead paint chips from abandoned buildings. Platt et al. (1999) document a similar wing droop in a Turkey Vulture (*Cathartes aura*), and this same symptom has been seen in other avian species.

Veterinarians and wildlife professionals are just beginning to investigate the potential effects of sublethal lead levels in animals, and the human lead poisoning literature serves as a wonderful resource to guide future research. Similarly, physicians and public health officials must also be willing to shift

their anthropocentric focus in order to fully identify lead exposure risks to humans. For example, there have been numerous case reports in which a child was tested and found to have elevated blood lead levels through a pet dog first being diagnosed with lead poisoning. Thomas et al. (1976) reported that a blood lead concentration of diagnostic significance in a family dog resulted in a six-fold increase in the probability of finding a child in the same family with similarly elevated blood lead levels. There is also a possible connection between lead in the tissues of waterfowl or game animals and human health. Sportsmen and their families may be exposed to high lead concentrations from shot residue in the meat of hunted waterfowl. Johansen et al. (2003) found that hunters who reported regularly eating birds hunted with lead shot had significantly higher mean blood lead levels than hunters who reported not eating hunted birds, 128 µg/L and 15 µg/L respectively. Even when bullet fragments are not present, secondary lead ingestion in waterfowl hunters can also occur through consuming the livers of chronically lead poisoned birds (Guitart et al. 2002).

OVERCOMING SPECIES ISOLATION

In addition to the separation that exists between the realms of human and animal health, lead poisoning research also tends to be compartmentalized within specific taxonomic groups. The research on lead toxicosis in wildlife concentrates predominantly on avian species. Most of the current literature falls within discrete categories such as raptors, loons, or waterfowl, and discusses one specific route of exposure. For example, literature concerning lead poisoning in raptor species such as Bald Eagles (*Haliaeetus leucocephalus*) and California Condors (*Gymnogyps californianus*) focuses primarily on ingestion of lead gunshot embedded in prey or scavenged carcasses (Janssen et al. 1986, Mateo et al. 2001). Studies of lead-related mortality in the Common Loon (*Gavia immer*) have identified ingestion of fishing sinkers as the prime route of lead exposure (Pokras and Chafel 1992, Sidor et al. 2003). In a wide variety of waterfowl species, toxicosis resulting from accidental intake of spent lead shot has been reported in the literature for over 100 years (Pain 1992).

Increased collaboration among researchers with expertise in different taxa is needed in order to advance our knowledge of lead in the environment. Approaching lead poisoning as just a waterfowl problem or just a raptor problem impedes progress toward effective policy changes. For instance, the impact of lead fragments remaining in carcasses and gut piles on condor populations has been thoroughly investigated, but we know almost nothing about the effects on other avian and mammalian scavengers. While it is crucial to comprehend the pathology within a particular species, we must also gain a better understanding of lead effects at the level of ecosystems. We know lead can accumulate in organisms such as invertebrates and plants, but we still have much to learn about how this influences the rest of the food chain (Pattee and Pain 2003). Deciphering the intricate web of environmental lead sources and exposure routes will allow us to implement better strategies to reduce the occurrence of lead poisoning in all species.

The Gray Squirrel (*Sciurus carolinensis*) may be a prime illustration of our need for a more comprehensive understanding of lead exposure sources in wildlife. Recently, one of these rodents was brought to the Tufts Wildlife Clinic and found to have a markedly elevated blood lead level of over 65µg/dL. Anecdotal stories of squirrels chewing on lead chimney flashing have been reported by homeowners for years, and recently a New Hampshire Fish and Game biologist confirmed that she regularly receives calls about "problem" squirrels that continually gnaw on chimney flashing. Analogous to children eating flecks of lead-based paint, this may be evidence of pica in squirrels. It may also be one explanation for the lead-poisoned predators like Red-tailed Hawks and Barred Owls that are periodically submitted to Tufts' Wildlife Clinic. Because millions of homes nationwide have lead flashing around chimneys, doors, and other openings, this appears to represents an overlooked source of plumbism in wildlife.

The literature on lead poisoning in non-human species is both broad and deep. Virtually all vertebrate taxa have been documented as experiencing lead poisoning (Table 4) although adults of some species appear to be more resistant than others. Lead toxicosis is well documented in mammals, from marine

Table 4. Species in which lead poisoning has been well documented.

people, other primates
many songbirds
loons
woodpeckers
hawks, eagles (Bald, Golden, others)
herons, flamingos, pelicans
vultures (including condors)
gulls
waterfowl (many species)
turkeys, quail, grouse, bob-white, pheasant
cranes, rails
reptiles (snapping turtle, crocodile, iguana)
parrots (many species)
squirrels, rabbits
woodcock, snipe
horses and cattle
Mourning Doves, pigeons
sheep and pigs
bats (micro and macrochiropterans)
dogs and cats
fish (many species)
rats and mice

species (Shlosberg et al. 1997, Zabka et al. 2006) to cattle and horses (Palacios et al. 2002, Sharpe and Livesey 2006) to bats (Skerratt et al. 1998, Bennett et al. 2003, Walker et al. 2007) and rodents. Useful reviews are provided by Priester and Hayes (1972), Humphreys (1991) and Mautino (1997).

A great many avian species have been shown to experience lead poisoning, in the wild and in captivity. In addition to waterfowl, raptors, loons, songbirds, and psittacines, many upland species, including gamebirds, are regularly poisoned by lead (Artman and Martin 1975, Kennedy et al. 1977, Locke and Friend 1992, Platt et al. 1999, Morner and Petersson 1999, Burger and Gochfeld 2000, Lewis et al. 2001, Mateo et al. 2001, Vyas et al. 2001, Scheuhammer 2003, Rodrigue 2005, Fisher et al. 2006).

Reptiles and amphibians also are affected by lead. Although the adults of some reptile species seem relatively resistant, early developmental stages do appear sensitive, especially among those amphibian species that have aquatic eggs and larvae that are in contact with the sediments (Barrett 1947, Kober and Cooper 1976, Overmann and Kracjicek 1995, Stansley and Roscoe 1996, Stansley et al. 1997, Borkowski 1997, Burger 1998, Rice et al. 1999, Vogiatzis and Loumbourdis 1999, Rice et al. 2002, Rosenberg et al. 2003, Arrieta et al. 2004, Mouchet et al. 2007).

The situation seems to be similar in fish. Adult fish of some species appear to be relatively insensitive to acute toxicity, but their eggs and larvae can show dramatic effects at low levels of exposure, sometimes resulting in population level effects and ecosystem alteration (Carpenter 1924a, b, Dilling et al. 1926, Jones 1964, Srivastava and Mishra 1979, Birge et al. 1979, Johansson-Sjöbeck and Larsson 1979, Newsome and Piron 1982, Hodson et al. 1984, Coughlan et al. 1986, Dallinger et al. 1987, Tewari et al. 1987, Eisler 1988, Tulasi et al. 1989, Sorensen 1991, Weber et al. 1997, Kasthuri and Chandran 1997, Chaurasia et al. 1996, Chaurasia and Kar 1999, Shafiq-ur-Rehman 2003, Martinez et al. 2004, Shah 2006). It seems clear that as more studies explore the sub-lethal effects of lead exposure in non-human species, there will be increased emphasis on integrating our thinking so that threats to human health are understood in the context of an over all environmental well-being.

Although it is not central to the present discussion, it is worth noting that there is abundant literature on lead in invertebrates. Some of this regards direct toxicity, but there is also literature on the ability of some invertebrates to accumulate lead (and other heavy metals) and to cause indirect toxicity to vertebrates that eat them (Grosell et al. 2006, Ma 1982, 1987, 1989, Scheuhammer 2003).

Similarly there is evidence that plants can also be affected by lead; either experiencing toxicity or as bioaccumulators (Malanchuk and Gruendling 1973, Manninen and Tanskanen 1993, Xiong 1998, Terry and Bañuelos 2000). Much remains to be learned about the effects of these processes on animal lead accumulation and health.

THE LIMITATIONS OF REGULATIONS

In April of 2005, the US Consumer Product Safety Commission announced a nationwide recall of 1.5 million children's fishing rods because it was found that the paint on the rod exceeded the 0.06% limit for lead. Parents were instructed to discontinue the use of the product immediately. At the same time,

an online retailer specializing in children's fishing gear was selling a product called "The Ultimate Fishing Kit for Kids." The kit comprised a plastic tackle box packed with 78 pure lead fishing sinkers. This and hundreds of other lead-stocked fishing kits designed for children are still widely available and have never been subject to a recall.

This example highlights the disjointed nature of current efforts to reduce lead exposure. Because many agencies in the USA regulate the various aspects of lead use—ranging from the Department of Labor for mining safety, to the Environmental Protection Agency for environmental pollution, to the Fish and Wildlife Service for the hunting of migratory birds—initiatives to limit lead's toxic effects have been myopic, lacking multidisciplinary perspective. In the USA, the banning of lead-based paint for residential use in 1978, the phasing-out of leaded fuel for on-road vehicles between 1973 and 1995, and most recently the mass recall of imported toys containing lead each represents an independent effort mobilized by separate groups. These measures have led to vast improvements in ecological health and helped protect human lives in much of the developed world. Annest et al. (1983) reported a 37% decrease in average blood lead levels in the USA between 1976 and 1980, associated with a reduction in the lead content of gasoline during this period. But this and other lead products are still widely available in developing countries. Lead is still a major component in some industrial paints, and leaded fuel continues to be sold for off-road uses such as aircraft, automobile racing, farm equipment, and marine engines. Even more alarming is the multiplicity of common household products that still contain lead – everything from curtain weights, solder, and batteries to imported ceramics, candy, and hair dyes.

Fortunately there is increased pressure to reduce lead in the USA and abroad. The European Union is engaged in efforts to eliminate several uses of lead. Groups in India, China, Australia, Nigeria and other countries are focused on eliminating leaded paints and gasoline and reducing human and animal exposure. Agencies concerned with the globalization of world economies and the internationalization of recycling (e.g., electronics) are increasingly taking steps to reduce toxic exports to developing countries in which protection for worker health may be lacking (Grossman 2006). Even in developed countries advocates for environmental justice have pointed out that exposure to lead and other toxic material falls disproportionately on many of the most vulnerable in our communities, often non-white populations with little education living in poorer neighborhoods (Bullard 1994). Clearly all stakeholders must be included in discussions of conservation and environmental health.

Although the toxicity of lead has been widely understood and reported for hundreds of years, progress to improve regulatory measures has been excruciatingly slow. Lead is cheap and easy to work with, and consequently the serious health risks associated with this metal are often overshadowed by its economic value. The continued prevalence of lead poisoning in both humans and animals is a signal that current policies are inadequate. A unified approach focused on interdisciplinary collaboration between specialists in human health, animal health, and ecological health is needed if we hope to make further progress in developing more protective legislation.

Like the classic story from the Far East of the five blind men and the elephant, each group of stakeholders perceives the lead (Pb) issue differently. Some will say, "What lead problem?" Others will say, "There may be a problem, but more research is needed." But for those of us who are convinced that it is necessary NOW to significantly decrease the quantities of this toxic metal that humans put into the environment, a number of approaches should be considered. These might include:

1. dramatically improving the marketing of non-toxic products,
2. exploring new business models, including the possibility of imposing taxes on Pb items (and perhaps investing those tax dollars in conservation), and/or providing tax and pricing incentives for non-toxic items,
3. improving educational efforts, especially those directed at sportsmen and their families,
4. developing new legislative approaches
5. encouraging technological innovation to find new non-toxic alternatives.

CONNECTING SCIENCE AND SPORT

The use of lead for hunting and fishing sports represents a particularly challenging situation. In the past 20 years, Vermont, New York, Maine, Massachusetts, New Hampshire, Great Britain, Denmark, and Canada have all passed legislation restricting the use of certain types of lead fishing gear. In 1991, lead shot was banned for use in hunting waterfowl in the United States. Most recently, California passed a bill in October 2007 banning the use of lead ammunition in areas of California Condor habitat. Unfortunately, these legislative initiatives designed to protect wildlife are often met with resistance from industry and sportsmen. Much of this opposition to proposed lead bans results from a lack of communication between scientists and the public. For example, many sportsmen's groups such as the US Sportsmen's Alliance condemn attempts to prohibit lead as an infringement on their rights, rather than a means to protect the health of people and wildlife. There is a misconception that lead prohibition laws are introduced by groups who oppose shooting and fishing sports solely as a tactic to limit these activities. Inaccurate information continues to plague a complete and successful transition to non-toxic hunting and fishing gear, and therefore establishing an open dialogue between researchers, sportsmen, and policy makers is critical.

Strict legislation banning the use of lead hunting and fishing gear that does not provide for the interests of sportsmen would result in ardent protest, low compliance, and ultimately would fail to resolve the lead poisoning issue. To bring an end to the problem once and for all, scientists and health professionals must find ways to better collaborate with hunting and fishing groups. We need to approach the issue in a way that encourages people to take a proactive role in eliminating this environmental crisis. Appealing to the conservationist roots of hunters and anglers is one way to do this. Many sportsmen are either unaware of the ecological damage caused by the use of lead gear or are skeptical of claims that a seemingly insignificant bullet or fishing weight could lead to such damaging effects for wildlife. It is therefore essential that we ramp up our efforts to reach out to sportsmen and educate them about the scientific rationale for moving away from lead, and to do so in a way that does not condemn their practices or their sport. In addition, working with manufacturers to provide sportsmen with more nontoxic alternatives that offer the same performance and practicality as lead is an important piece of a successful lead phase out. This cooperative approach will allow sportsmen to play a positive role in efforts to limit the use of lead, and in the end is the key to a permanent solution.

BREAKING DOWN THE BARRIERS

The effects of lead poisoning are not confined to human health nor to any one species of animal. Thus, we will never successfully gain control of the problem unless we take an approach that is all-inclusive. We cannot continue to view the different aspects of plumbism in isolation from one another. Paint, gasoline, occupational exposure, toys, bullets, fishing gear, and all the other sources of lead are not separate issues but rather are components of the same fundamental problem.

Developing strategies to achieve better integration among conservation and health disciplines will broaden our scientific understanding of lead poisoning and accelerate progress toward solutions. Currently, studies on lead poisoning in people, wildlife, and domestic animals are all published separately in journals devoted to those specific fields. Establishing resources that include lead poisoning literature from all domains will promote a better flow of ideas and scientific knowledge between disciplines and allow researchers to see the interconnectedness of human and animal plumbism. Conferences and meetings that address multiple aspects of lead poisoning provide a prime opportunity for researchers and action groups to network with experts in different disciplines. The EPA's National Lead Poisoning Prevention Week in October concentrates primarily on childhood poisoning from lead paint, but would be an ideal opportunity to spread public awareness and increase communication about all of the other issues associated with lead poisoning. Bringing together a wide range of stakeholders to participate in the lead poisoning dialogue will allow us to find solutions that are scientifically accurate, environmentally sound, economically viable, and socially acceptable.

An increasing number of organizations are now realizing the value of a conservation medicine-based approach to the lead poisoning issue and are making efforts to break down disciplinary barriers. Several groups such as the EPA's Leadnet, The Lead Education and Abatement Design Group in Australia, and the Tufts Veterinary School's Lead and Health group have formed listserves and contact databases to facilitate communications among people—diverse fields such as environmentalists, sportsmen, veterinarians, wildlife professionals, lead industry representatives, citizen action groups, and environmental justice groups. The Midwest Fish and Wildlife Conference held in December 2007 included a session on lead poisoning that was attended by public health professionals, veterinarians, wildlife biologists, sportsmen, and lead fishing tackle manufacturers. The Peregrine Fund's 2008 Lead Ammunition Conference focused on implications for both human and animal health. Finally, a session has been proposed for the EcoHealth II conference in December 2008 that would bring together a wide range of professionals to focus on lead and its many health and environmental effects.

CONCLUSIONS

Despite the fact that lead poisoning is well understood, it still threatens the health of millions of people, domestic animals and wildlife worldwide. Barriers among disciplines have impeded the scientific understanding of lead toxicosis and slowed policy initiatives to protect the health of animals and people. A conservation medicine-based approach focusing on increasing collaboration among professionals working in different fields offers the best hope for understanding and eliminating this ancient problem.

ACKNOWLEDGEMENTS

We gratefully acknowledge Springer Publishers, and The International Association for Ecology and Health and their journal EcoHealth for allowing us to utilize portions of a previously published article (Pokras and Kneeland 2008) in the current paper. We would like to thank the many veterinary students and volunteers whose help over the years has made our studies possible. We thank Mr. R. Wood, Dr. J. McIntyre of Utica College (Syracuse University), Dr. P. Spitzer of the Center for Northern Studies, and Mr. A. Major and Ms. C. Perry of the US Fish and Wildlife Service for their invaluable advice and assistance. We thank Drs. M. Friend and J. C. Franson of the National Wildlife Health Research Center (USGS), Dr. R. Haebler of the Atlantic Ecology Division (US EPA) and Dr. E. Silbergeld for their wise advice and encouragement over the years. The Pathology Department of Tufts Cummings School of Veterinary Medicine (TCSVM) generously contributed their expertise to this project.

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